ENDOVASCULAR AND SURGICAL TECHNIQUES

A New Therapeutic Approach to Popliteal Artery Entrapment Syndrome (PAES)

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Objectives: An alternative therapeutic approach to popliteal artery entrapment syndrome (PAES) with vascular complications.

Materials: Three male patients (16, 42 and 68 years) with thrombotic and/or embolic obstructions of popliteal or crural vessels as a consequence of popliteal entrapment syndrome.

Methods: Combined catheter treatment consisting of percutaneous transluminal thrombectomy (PTEE), local thrombolysis (LTL) and percutaneous transluminal dilatation (PTA) was performed for thrombotic and embolic obstructions of popliteal or crural vessels. The aberrant tendonmuscular structures were surgically corrected at a later date.

Results: After the procedure systolic ankle/arm pressure ratios in the three patients improved from 0.66 to 1.13, 0.57 to 1.07 and 0.46 to 1.10. Twelve, 8 and 4 months later the patients were asymptomatic. None of the patients showed popliteal artery compression during plantar flexion on Duplex scanning.

Conclusions: This new therapeutic approach avoids direct vascular surgery with bypass or patch implantation in patients with PAES who are often young. The long term results remain to be evaluated.

Key Words: Popliteal entrapment syndrome; Percutaneous transluminal thrombolysis; Thrombectomy.

Introduction

Intermittent claudication in young patients is most often caused by premature atherosclerosis and occasionally by arterial embolism, cystic disease of the adventitia or fibromuscular hyperplasia. In less than 1% of all patients with peripheral arterial occlusive disease popliteal artery entrapment syndrome (PAES) is the cause.¹,² PAES is caused by an anomalous relationship between popliteal artery and its surrounding muscular and/or tendinous structures. Repetitive traumatisation by aberrant muscles or tendons damages the artery, and may result in thrombotic occlusion. Embolisation into the calf arteries may also occur. The conventional treatment consists of surgical revascularisation (thrombendarterectomy, bypass, vein patch graft) of the occluded artery and/or release of the entrapped vessel.³

We report on three patients with PAES with thrombotic and/or embolic complications treated by a new approach. Initially, the occluded popliteal and crural arteries were reopened by a combination of local thrombolysis (LTL), percutaneous transluminal thrombectomy (PTEE) and percutaneous transluminal dilatation (PTA) with a balloon catheter.⁴⁻⁶ Surgical correction of the muscular or tendinous anomaly causing the arterial entrapment was performed at a later date.

Technique

Under local anaesthesia, the common femoral artery was punctured in an antegrade direction and an introducing sheath (6 F) inserted using the Seldinger
technique and 5000 IU of heparin given intra-arterially. Under fluoroscopic control, a steerable guide wire was passed through the occlusion and an endhole aspiration catheter was advanced into the proximal part of the thrombus or embolus. After removal of the guide wire, negative pressure was established by means of a 50 ml syringe and the thrombotic and/or embolic material was sucked into the syringe or trapped at the tip of the catheter which was then carefully retracted to remove the clot. For clot aspiration from the calf arteries, a 5 F aspiration catheter was used. In two patients thrombotic and/or embolic material was first infiltrated with urokinase at a dosage of 200,000 IU and 300,000 IU, respectively. After partial lysis the remaining material was aspirated. Percutaneous transluminal angioplasty of any apparent stenosis was performed.

After the procedure heparin was administered intravenously at a dosage of 625 to 800 IU/h for 48 h and oral anticoagulation was started for 3 months followed by aspirin 250 mg/day. Surgical correction was performed 3–6 weeks after catheter treatment.

Case Reports

Patient 1

A 16-year-old football player complained of a 1 year history of increasing coldness, numbness and burning pain in the right foot during exercise. Physical examination revealed absent foot pulses at the right lower limb. Systolic ankle/arm pressure ratios were 0.66 on the right compared to 1.08, on the left. Arteriography revealed an occlusion of the distal popliteal artery and proximal tibial arteries (Fig. 1a). Duplex scanning of the popliteal fossa demonstrated complete compression proximal to the occluded part of the popliteal artery during plantar and dorsal flexion of the foot. At the site of compression the vessel was moderately dilated with a diameter of 0.8 cm. Magnetic resonance imaging documented an anomalous course of the medial head of the gastrocnemius muscle (Fig. 2). The popliteal artery, tibio-peroneal trunk and the posterior tibial artery were recanalised by PTEE (6 F introducing sheath, 5 F aspiration catheter) and LTL with 200,000 IU of urokinase administered into the thrombotic occlusion (Fig. 1b). One day after catheter treatment the systolic arm/ankle ratio had risen to 0.9. 625 IU/h of heparin was given intravenously for two days. Aspirin (250 mg/day) was subsequently used instead of oral anticoagulation because the patient continued to play football.

Three weeks after successful catheter treatment, myotomy of the medial head of the gastrocnemius muscle was performed. Intraoperatively it was found that the popliteal artery was running through the medial head of the gastrocnemius muscle. Four months after the operation the patient was asymptomatic with a systolic ankle/arm pressure ratio of 1.13.

Patient 2

A 42-year-old mountain hiker without cardiovascular risk factors complained of deteriorating claudication of the right foot during climbing for 6 months. Physical examination revealed absent pulses in the right foot and the systolic ankle/arm ratio was 0.57 compared to 1.09 on the left. Colour Duplex scanning of the popliteal artery demonstrated a significant stenosis of the distal part of the popliteal artery. With maximal plantar flexion of the foot popliteal artery was completely compressed at the site of the stenosis. Partially occluding thrombi were visualised in the proximal segments of all three calf arteries. Magnetic resonance imaging demonstrated an anomalous insertion of the medial head of the gastrocnemius muscle into the lateral femoral condyle. The patient underwent LTL with 300,000 IU urokinase and PTEE. (6 F introducing sheath, 5 and 6 F aspiration catheters). The peroneal and posterior tibial arteries were recanalised without complications. The stenosis of the popliteal artery was dilated with a 4 mm balloon catheter. Heparin was administered for 2 days intravenously at a dosage of 800 IU/h and oral anticoagulation was initiated for 3 months. Six weeks after catheter intervention, surgery was performed. Intraoperatively, the results of the MRI study were confirmed and the aberrant medial head of the gastrocnemius muscle into the lateral femoral condyle. The patient underwent LTL with 300,000 IU urokinase and PTEE. (6 F introducing sheath, 5 and 6 F aspiration catheters). The peroneal and posterior tibial arteries were recanalised without complications. The stenosis of the popliteal artery was dilated with a 4 mm balloon catheter. Heparin was administered for 2 days intravenously at a dosage of 800 IU/h and oral anticoagulation was initiated for 3 months. Six weeks after catheter intervention, surgery was performed. Intraoperatively, the results of the MRI study were confirmed and the aberrant medial head of the gastrocnemius muscle into the lateral femoral condyle. At a 1 year follow-up the patient was asymptomatic with a systolic ankle/arm ratio of 1.07. Colour Duplex showed no compression of the popliteal artery on maximal plantar flexion.

Patient 3

A 68-year-old man with a history of ischaemic heart disease, smoking and hypercholesterolaemia had been...
treated three times by local thrombolysis, percutaneous thrombembolectomy and percutaneous transluminal angioplasty because of calf claudication of his right leg caused by thrombotic occlusion of the popliteal and proximal calf arteries. On all three occasions the occluded arteries were successfully recanalised by the combined catheter intervention. Three months after the last procedure he was hospitalised again because of recurrent intermittent claudication. Physical examination revealed absent right foot pulses with a systolic ankle/arm pressure ratio of 0.46 compared to 1.05 on the left. Arteriography demonstrated atherosclerotic plaques in the superficial femoral artery and thrombotic occlusion of the popliteal artery and the tibioperoneal trunk (Fig. 3a). Percutaneous thrombembolectomy (PTEE) was successfully performed with a 6 F aspiration catheter (Fig. 3b). 800 IU/h of heparin was given for two days intravenously and oral anticoagulation was started. PAES was suspected because of the recurrent popliteal occlusions. Colour Duplex scanning documented complete compression of the popliteal artery during maximal plantar flexion of the foot. Computed tomography showed a displacement of popliteal artery and vein by an anomalous course of the medial head of the gastrocnemius muscle.

The anomaly was confirmed during surgery 4 weeks after catheter treatment. A small part of the medial head of the muscle was resected and the remaining part reinserted into the medial condyle. Eight months after the operation the patient was asymptomatic. Colour Duplex scanning demonstrated no popliteal artery compression during plantar flexion and the systolic ankle/arm ratio was 1.10.
Fig. 2. Magnetic resonance imaging of the popliteal fossa of patient 1. On the right side (R) the popliteal artery (1) and vein (3) are displaced by an anomalous course of the medial head of the gastrocnemius muscle (2). The left side shows normal anatomy.

Discussion

In patients 1 and 2 PAES was suspected because of young age, lack of cardiovascular risk factors and clinical findings. By contrast, in patient 3, advanced age and the presence of classical cardiovascular risk factors suggested arteriosclerotic disease as the cause of popliteal obstruction. The presence of PAES in this patient was suspected only after recurrent thrombotic occlusions of the vessel. In all 3 patients PAES was demonstrated by colour Duplex scanning with plantar flexion of the foot. Although the value of Duplex scanning is not well established, in our experience the technique seems to be accurate for the non-invasive diagnosis of PAES. Duplex scanning may even detect the condition in the presence of an occluded popliteal artery (patient 1). Magnetic resonance imaging or computed tomography of the popliteal fossa is recommended for delineating the underlying tendo-muscular anomaly. Compared to CT, MRI permits multiplanar images and provides a higher soft tissue contrast which may facilitate delineation of tendomuscular structures.

Medial displacement of the popliteal artery may be depicted by conventional arteriography, but is lacking in some of the more than 10 forms of popliteal entrapment. Chronic traumatisation of the popliteal artery by crossing tendons or muscles with anomalous insertion results in vascular damage and ultimately may lead to vessel occlusion. In about 10%
of the patients\(^3\) embolism occurs as was the case in patients 1 and 2. The principles of treatment of PAES with popliteal artery occlusion and/or crural emboli are to release the entrapped vessel and to restore normal blood flow to the limb. This is conventionally achieved by myotomy or resection of the underlying musculo-tendinous anomaly and by autogenous vein patch grafting or bypass.\(^3,14,16\) By contrast we used a combined strategy consisting of PTEE, LTL and PTA when necessary, followed by surgical correction of the anomalous tendomuscular structures at a later date. The obvious advantage of recanalisation of the native popliteal artery by percutaneous catheter treatment is that it avoids venous bypass in these patients who are often young. Steerability of the catheter systems under fluoroscopic control allows removal of thrombotic or embolic material not only from the popliteal artery but also selective clot aspiration from the tibial vessels.\(^4-6\) In our experience even partially organised embolic occlusions of the calf arteries may be safely removed with PTEE especially if LTL is also used.

In cases where the popliteal artery is extensively damaged with formation of an aneurysm surgical repair of the vessel segment is clearly indicated.\(^17\) A moderate dilatation of the popliteal artery was present in the third patient. However, since the maximal diameter was only 0.8 cm we decided to treat the patient by the combined approach. Although catheter treatment was successfully performed, long-term follow-up will show whether a popliteal artery aneurysm develops.

In our patients the time interval between catheter treatment and surgery varied between 3 to 6 weeks. We do not know the optimal time interval between catheter treatment and surgery. One may argue that delayed surgery jeopardizes catheter treatment. On the other hand the surgeon may prefer a delayed approach to prove the durability of successful catheter treatment.

Long-term follow-up data of patients treated by the conventional operative approach are very limited. In a study of 11 patients without vascular complications the sole repair of the anomalous tendomuscular structures resulted in a patency rate of the popliteal artery of 94% after a mean follow-up of 46 months.\(^3\) In 12 cases where vascular reconstruction was necessary the patency rate after a mean follow-up of 43 months dropped to 58% suggesting an unfavourable prognosis in these young active people.\(^3\) On the basis of these data our modified treatment procedure that preserves the native popliteal artery may result in a better prognosis. The procedure proved successful in the three patients described with continued patency after 1 year, 8 and 4 months respectively based on absence of intermittent claudication and ankle/arm pressure indices of more than 1.0. However, long-term results in respect to aneurysm formation and vessel patency have to be evaluated.

References


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